Progress, Prospects, and Research Needs on the Health Effects of Acid Aerosols

by Morton Lippmann*

Research on human exposure to acidic aerosols and the health effects of such exposures has substantially strengthened the hypothesis that such aerosols are a causal factor for excesses in human mortality and morbidity that have been previously associated with crude exposure indices such as British Smoke, total suspended particulate matter, and sulfur dioxide. Research reported at this symposium also showed that combined exposures to acid aerosols and other ubiquitous air pollutants such as O₃, NO₂, HNO₃, and SO₂ produce greater effects in both humans and animals than exposures to each agent separately. The responses reported ranged from physiological functions to lung structure. Furthermore, some of the effects were cumulative with increasing duration of daily exposure and number of repetitive exposures. Critical areas for further research include better definition of the critical temporal parameters affecting exposure and response, effects of mixed pollutant exposures, and pathogenetic mechanisms for acid aerosol-induced chronic lung damage.

Introduction

The reports of the four session rapporteurs, Mueller (1), Shy (2), Graham (3), and Folinsbee (4), and the paper by Albert on approaches to risk assessment for acid aerosols (5) provide ample evidence that our knowledge base on the health effects of acid aerosols has expanded considerably in the past 3 years. Looking back on my summary of major issues concerning exposures to airborne acidity and their human health effects from my rapporteur's report at the 1984 symposium (6), which was reproduced in my introductory paper at this symposium, I conclude that the summary judgments of 1984 were basically sound, but are now much more firmly established. The table in that paper could be updated by changes in about half a dozen entries, mostly by inserting somewhat lower effective concentrations and extending the time periods for effective exposures and effects.

In this paper, I attempt to provide an integrated summary of our current understanding of the health effects of acid aerosols. While the views expressed are consistent with the summaries and interpretive remarks of the four session rapporteurs, I have not attempted to summarize their reports. Rather, I have emphasized my overall impressions of the most significant of the new findings and their implications to an overall judgment of the public health implications of exposures to acid aerosols and to the needs for additional research.

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Symposium Summary

The subtitle of this symposium, "Addressing Obstacles in an Emerging Data Base," was an apt description of the content of the symposium. There is indeed an emerging data base in each of the four topic areas covered, i.e., (a) extent of human exposures to ambient acid aerosols; (b) epidemiologic studies of the health effects resulting from inhalation exposures; (c) studies of responses in experimental animals and model systems; and (d) studies of functional responses in humans to short-term inhalation exposures, both as fogs and as the more typical fine fraction aerosol. In all of these areas we have seen a significant expansion in our knowledge and understanding in comparison with those at the close of the previous international symposium on this topic at NIEHS in 1984 (6).

A good indication of the increasing maturity of the field was evident in the presentations at the closing session by the four rapporteurs. In summarizing and interpreting the papers presented in their sessions, each of them made reference to papers presented in other sessions as well. The free and open discussions throughout the meeting, especially those relating to mechanisms of biological action and pollutant interaction, were particularly valuable. As a result of these discussions, the research needs are much clearer, and bases for further collaborative studies among physiologists, toxicologists, epidemiologists, and exposure specialists have been established.

Among the notable research findings reported at this symposium were the first sets of continuous acid aerosol concentration data over extended periods of time in 204 M. LIPPMANN

U.S. cities by Spengler et al. (7); evidence for significant excesses in respiratory mortality and morbidity in West Germany from an acidic pollution episode in January 1985 by Wichmann (8), to complement the recent report by Dassen et al. of persistent decrements in lung function among Dutch schoolchildren during and following the same pollution episode (9); and evidence that decrements in lung clearance function are dependent on the duration of exposure as well as on concentration, both in rabbits by Schlesinger (10), and in humans by Spektor et al. (11). Other studies provided evidence that combined exposures to acidic aerosols and pollutant gases such as ozone (O_3) (12,13), nitrogen dioxide (NO_2) (10,12,13), nitric acid (HNO_3) , (14) and sulfur dioxide (SO_2) (14,15) produce greater effects than either alone. Such synergisms were seen for effects ranging from functional changes in respiratory mechanics by Koenig et al. (14) and Amdur and Chen (15), and particle clearance by Schlesinger (10), to changes in lung biochemistry by Last (12) and the size of lung lesions by Kleinman et al. (13). The various changes occurred in a variety of species, including rats (12,13), rabbits (10), and humans (14). Exposures of guinea pigs by Amdur and Chen (15) to a mixture of SO₂ and ultrafine metal oxide particles coated with sulfuric acid (H₂SO₄) produced persistent lung functional and structural changes following a single 3-hr exposure at 50 µg/m³ H₂SO₄, and cumulative changes after 5 exposure days at 20 µg/m³. These changes were comparable to those produced by pure H_2SO_4 at 200 $\mu g/m^3$ and those produced by single exposures to O₃. In another study reported by Gearhart and Schlesinger (16), rabbits exposed for 1 hr/day, 5 days/week, for 1 year at 250 μg/m³ H₂SO₄ showed persistent alterations in the rates of mucociliary and alveolar particle clearance, airway reactivity, airway caliber, and the density of secretory cells in the airways. The progression of these effects, and their similarity to those seen in human cigarette smokers, lends credence to a possible causal association between exposures to acid aerosols and the development of chronic bronchitis (17).

One paper in the symposium by Ito and Thurston (18) compiled previously unreported daily ambient acid aerosol concentration data in 1963 through 1971 winters for London, England, while a second paper by Thurston et al. (19) showed that these concentrations correlated more closely with total daily mortality than did the other available pollutant variables. Chronic bronchitis mortality rates were exceptionally high in London in earlier years, when acid aerosol concentrations were much higher than those after 1963. If acid-coated particles are indeed more toxic than pure acid droplets, it may account for some of the excess mortality and morbidity in London in earlier decades and provide a basis for extra concern about exposures to primary acidic aerosols downwind of smelters and power plants.

In summary, it is clearer than ever that H₂SO₄ in ambient aerosols is a highly likely causal factor for

effects on lung structure and function. Furthermore, evidence is rapidly accumulating that H2SO4 and other ubiquitous air pollutants (O₃, NO_x, SO₂) can potentiate each others effects. The statistically significant associations reported by Bates (20) between daily sulfate aerosol concentrations and daily hospital admissions for respiratory diseases in Ontario from 1978 to 1985 and between annual average sulfate concentrations and total annual mortality in 98 U.S. communities in 1980 reported by Ozkavnak and Thurston (21) may be due to the actions of acidic aerosols as potentiated by one or more of the common pollutant vapors. Questions such as these make it especially important to encourage more basic science. In that regard, the papers presented at this symposium on the influences of the chemical and physical forms of ambient air acids on airway doses and responses by Larson (22), and on the effects of inhaled acids on airway mucus by Holma (23), were particularly valuable and were repeatedly cited in the discussions of the papers that focused primarily on exposure-response relationships.

The amount of progress made in the last 3 years is remarkable in view of the very limited number of research groups that are active in this area. The remaining obstacles in the emerging data base include the still extremely limited data base on the nature and extent of human exposure, the nature of the interactions between the acidic and oxidant pollutants in the ambient air, and their sequential and interactive effects on the respiratory tract after their deposition and uptake, and the ways in which pollutant-induced responses in lung defenses can contribute to the pathogenesis of lung disease.

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